Health Effects of Acid Aerosols on North American Children: Respiratory Symptoms

Douglas W. Dockery,[1,2] Joan Cunningham,[1] Andrew I. Damokosh,[1] Lucas M. Neas,[1,2,3] John D. Spengler,[1] Petros Koutrakis,[1] James H. Ware,[4] Mark Raizenne,[1,5] and Frank E. Speizer [1,2]

[1]Department of Environmental Health, Harvard School of Public Health, [2]Channing Laboratory, Brigham and Women's Hospital, Harvard Medical School, [3]Department of Epidemiology, Harvard School of Public Health, and [4]Department of Biostatistics, Harvard School of Public Health, Boston, MA 02115 USA; [5]Environmental Health Directorate, Health Canada, Ottawa, K1A0L2 Canada

We examined the respiratory health effects of exposure to acidic air pollution among 13,369 white children 8 to 12 years old from 24 communities in the United States and Canada between 1988 and 1991. Each child's parent or guardian completed a questionnaire. Air quality and meteorology were measured in each community for a 1-year period. We used a two-stage logistic regression model to analyze the data, adjusting for the potential confounding effects of sex, history of allergies, parental asthma, parental education, and current smoking in the home. Children living in the community with the highest levels of particle strong acidity were significantly more likely [odds ratio (OR) = 1.66; 95% confidence interval (CI) 1.11–2.48] to report at least one episode of bronchitis in the past year compared to children living in the least-polluted community. Fine particulate sulfate was also associated with higher reporting of bronchitis (OR = 1.65; 95% CI 1.12–2.42). No other respiratory symptoms were significantly higher in association with any of the air pollutants of interest. No sensitive subgroups were identified. Reported bronchitis, but neither asthma, wheeze, cough, nor phlegm, were associated with levels of particle strong acidity for these children living in a nonurban environment. *Key words:* acid aerosols, air pollution, bronchitis, children, respiratory symptoms. *Environ Health Perspect* 104:500–505(1996)

Although there has been much public and scientific debate about acid rain and its precursors, the focus in the past was largely on ecological effects and secondary health effects related to mobilization of toxic metals. Concern about the possible adverse human health effects resulting from exposure to acid aerosols has been more recent (*1,2*).

Experimental studies of sulfur dioxide and total suspended particulates have shown adverse health effects in both animals (*3*) and human subjects (*4*). In epidemiologic studies, particulate pollution has been associated with decrements in lung function (*5*), higher prevalence of respiratory symptoms and illness (*6–8*), and higher rates of hospitalizations (*9,10*) and mortality (*11*), even at the levels currently found in many urban areas.

Fine particles, which penetrate deeply into the lungs, are produced by direct emissions from combustion sources or atmospheric reactions of gaseous pollutants. These fine particles are often very acidic. The observed health effects associated with particulate pollution may be attributable at least in part to the acidity of the fine particles (*12,13*). Previous population-based studies lacked direct measures of ambient aerosol acidity, but recent improvements in atmospheric monitoring techniques have made it possible to directly measure particle strong acidity (*14*).

In children from the Six Cities Study, reported bronchitis and chronic cough were associated with total suspended particles (*7*) and with particulate matter less than 15 μm in aerodynamic diameter (*8*). In a later analysis of children from the same six communities plus Kanawha County, West Virginia (*15*), the city-specific summertime mean of particle strong acidity was associated with significantly higher risk of bronchitis and chronic cough.

This study was specifically designed to examine the relationship between long-term, intermittent exposure to particle strong acidity and the respiratory health of children (*16*). In this article, we describe the associations between particle strong acidity and respiratory symptoms.

Methods

Air monitoring. We selected 24 communities on the basis of previously measured sulfate and ozone concentrations and demographic characteristics (*17*). The study included 18 sites in the United States and 6 sites in Canada to provide a wide range of expected particle strong acidity and ozone levels. Communities were predominantly suburban or rural with homogeneous, relatively stable populations and no major local sources of air pollution.

Air pollution measurement methods and calibration procedures are described in an accompanying paper (*17*). Air pollution monitoring began at each site before the summer high acid–ozone exposure period and continued for at least 11 months. Particulate pollutants were sampled for 24 hr every other day. Inhalable particulate matter with an aerodynamic diameter >10 μm (PM_{10}) was sampled using a Harvard Impactor (*18*). Fine particulate matter with an aerodynamic diameter >2.1 μm ($PM_{2.1}$) was sampled using a glass impactor/filter pack system. Fine particle strong acidity, fine particle sulfate, and gaseous acids (nitrous and nitric acids) were sampled using the Harvard EPA Annular Denuder System (HEADS) in 21 communities (*19–21*). In three communities with expected low levels of gas-phase acidity, the Harvard Impactors with ammonia denuders were used. Strong acidity was measured by pH analysis (*20*) and sulfate was measured by ion chromatography of the extracted particles. Ozone was monitored continuously.

We grouped the 24 communities by the sources of particulate mass and acidity (*17*). The Sulfate Belt was defined as the 11 sites with high sulfate and acidity concentrations lying generally along the Appalachian Plateau and the Allegheny Mountains. The Transport Region included communities north and east of the Sulfate Belt, which are generally downwind of the principal sulfur emissions in the United States. The remaining commu-

Address correspondence to D.W. Dockery, Environmental Epidemiology Program, Department of Environmental Health, Harvard School of Public Health, 665 Huntington Avenue, Boston, MA 02115 USA.

We thank C. Franklin, D. Villeneuve, and R. Burnett for their advice and support. This study was possible because of the hard work of M. Fay, A. Jemas, E. Donahue, C. Fischer, S. Hanig, L. Kern, J. L. McClelland, C. Panek, B. Phelps, E. Solomon, J. Mortimer, K. Wilby, and J. Moore, E. Graham, E. Haynes, M. Ramsey, the air monitoring staff at the Harvard School of Public Health, and the field staff at the various locations. We thank the school administrators, teachers, and especially the parents and children in the participating communities. This paper was presented in part at an international conference sponsored by the American Thoracic Society in San Francisco, California, 18 May 1993. This study was supported in part by National Institute of Environmental Health Science grants ES-04595 and ES-0002, U.S. Environmental Protection Agency cooperative agreement CR 816071, and Health Canada. L. M. N. and J. C. were supported in part by a National Institute of Health National Research Service Award (HL-07427).
Received 21 August 1995; accepted 22 January 1996.

nities were grouped as West Coast (three sites) and Background (four sites).

Respiratory health questionnaire. The study protocol and methods were approved by the Institutional Review Board at the Harvard School of Public Health and also by the local health department and school board(s) in each community. We contacted principals of selected schools directly and invited them to participate. In communities with fewer than 750 children in the fourth and fifth grades, all schools with at least 10 students in each grade were approached. In larger communities, selection of schools was based either on proximity to the air monitoring station or on random selection, depending on the geography of the school district(s). We distributed standard respiratory health questionnaires to the children at participating schools in September and October of 1988 (eight communities), 1989 (nine communities), and 1990 (seven communities). Children were instructed to have the questionnaire completed by a parent or guardian and to return it to school the following day. Classroom (but not individual) incentives were used to encourage participation, and questionnaires were distributed multiple times over several weeks. The overall response rate (returned questionnaires plus written refusals) was 91%, with a range of 85–99% for the individual communities (Table 1).

Later in the school year, each school was visited again and the pulmonary function of children who had their parents' permission to participate was measured. The results of the analysis of pulmonary function are reported in an accompanying article (*23*).

Definition of respiratory symptoms. Children's respiratory symptoms in the previous year were determined from questionnaire responses defined as follows: 1) asthma: ever diagnosed with asthma by a physician plus asthmatic symptoms present in the past year; 2) attacks of wheezing: episodes of shortness of breath with wheezing and/or episodes of wheezing after playing hard or exercising; 3) persistent wheeze: wheeze apart from colds and/or wheeze for 3 or more days out of the week for a month or longer; 4) chronic cough: cough first thing in the morning for as long as 3 months in a row and/or cough at other times during the day or night for as long as 3 months in a row; 5) chronic phlegm: congestion in the chest or bringing up phlegm other than with colds for as long as 3 months in a row; 6) bronchitis: a chest illness diagnosed as bronchitis; 7) asthmatic symptoms: asthma, attacks of wheezing, and/or persistent wheeze; and 8) bronchitic symptoms: chronic cough, chronic phlegm, and/or bronchitis. Only reports of symptoms in the past 12 months were considered.

Statistical methods. Previously reported analyses of city-specific health outcomes have demonstrated a larger between-city variation than would be predicted by interindividual variation (*7,8*). In the present study, we used a two-step analysis to correct for any excess between-city variability. In the first step, city-specific–adjusted logits of symptom prevalence were calculated from a logistic regression model for the respiratory symptom of interest controlling for sex, child's history of diagnosed allergies, parental history of diagnosed asthma (for either biological parent), parental post-high school education (for either parent), and current smoking in the home. In the second step, these city-specific–adjusted logits were regressed against the city-specific annual mean air pollution concentrations using weights inversely proportional to the sum of the between-city and within-city variances of the adjusted logits. Analyses were done using the SAS software (SAS Institute, Inc., Cary, North Carolina). We scaled the regression coefficients from the second step to the range of the city-specific means for the air pollutant of interest (highest to lowest; Table 1) and expressed them as odds ratios (OR) and 95% confidence intervals (95% CI).

To assess the consistency of the evidence and to search for sensitive subgroups, the analyses were repeated stratifying for sex; history of a severe chest illness before the age of 2; use of a humidifier in the home; lung function in the lowest quartile of forced vital capacity (FVC); current exposure to environmental tobacco smoke at home; exposure to maternal smoking during pregnancy; year of study; country; and eastern versus western cities.

Results

The city-specific ranges in the annual pollutant means (Table 1) were 51.9 nmol/m^3 for particle strong acidity, 6.8 μg/m^3 for sulfate particles, 14.9 μg/m^3 for respirable particulate matter ($PM_{2.1}$), and 17.3 μg/m^3 for inhalable particulate matter (PM_{10}). The gaseous acids (HNO_2 and HNO_3) were measured in all but three communities and together had a range of 111 nmol/m^3. City-specific mean particle strong acidity was moderately correlated with PM_{10} [Pearson's correlation coefficient (r) = 0.47], strongly correlated with sulfate (r = 0.90) and $PM_{2.1}$ (r = 0.82), and essentially uncorrelated with gaseous acids (r = 0.07). Three exposure parameters were considered for the annual mean ozone concentration: the average maximum 1-hr mean (range 46 ppb), the average daytime 8-hr (1000–1800 hr local time) mean (range 39 ppb), and the average daily 24-hr mean (range 19 ppb). All three ozone parameters were highly correlated across the 24 communities with Pearson correlation coefficients ranging from 0.74 to 0.98. By design, city-specific mean particle strong acidity was only weakly correlated with the three ozone parameters; the strongest correlation was with the average 1-hr maximum ozone concentration (r = 0.37).

A total of 17,008 children were eligible to participate in the study, of whom 15,523 children (91%) returned a questionnaire. Children who were reported to have cystic fibrosis, a history of a chest

Table 1. Summary of city-specific annual mean air pollution concentrations: 24 communities, United States and Canada, 1988–1991

Pollutant	Grand mean	SD	Minimum	Maximum	Range
Particulate matter					
Inhalable (μg/m^3)	23.8	5.0	15.4	32.7	17.3
Respirable (μg/m^3)	14.5	4.2	5.8	20.7	14.9
Sulfate particles (μg/m^3)[a]	4.7	2.2	0.7	7.4	6.8
Particle strong acidity (nmol/m^3)	27.5	16.2	0.0	51.9	51.9
Gaseous pollutants					
Ozone, 1-hr max. (ppb)	46.5	8.5	26.9	72.5	45.6
Ozone, 8-hr daytime avg. (ppb)	38.4	7.5	21.0	60.4	39.4
Ozone, 24-hr avg. (ppb)	27.8	5.1	16.3	34.8	18.5
Sulfur dioxide (ppb)[b]	4.8	3.5	0.2	12.9	12.7
Ammonia (ppb)[b]	1.3	1.4	0.1	5.8	5.7
Nitrous acid (ppb)[b,c]	0.6	0.4	0.1	1.4	1.3
Nitric acid (ppb)[b,c]	0.9	0.4	0.3	2.1	2.7
Acidity[b]					
Particle strong acidity (nmol/m^3)	31.1	14.0	8.5	51.9	43.4
Gaseous acids (nmol/m^3)[d]	59.4	28.4	22.7	134.0	111.3
Total (nmol/m^3)	90.6	33.9	38.0	150.5	112.5

[a]1 μg/m^3 = 10.4 nmol/m^3.
[b]Excluding Egbert, Ontario; Yorkton, Saskatchewan; and Aberdeen, South Dakota.
[c]1 ppb = 40.9 nmol/m^3.
[d]Gaseous acids = nitrous + nitric acid.

Table 2. Distribution of covariates used in the respiratory symptoms model by community: 13,369 children, 24 comunities, United States and Canada, 1988–1991

	Site code	Participation (%)	No. of children		Females (%)	Allergy history (%)	Parental asthma (%)	Parental post-high school education (%)	Current smoking in the home (%)
			Excluded	Included					
Sulfate Belt									
Hendersonville, TN	HEN	85	59	504	56	40	13	68	55
Oak Ridge, TN	OAK	96	119	540	46	41	15	83	42
Morehead, KY	MOR	93	48	393	50	30	14	52	61
Blacksburg, VA	BLB	94	124	666	48	37	16	74	37
Charlottesville, VA	CHV	91	171	522	46	24	12	45	53
Zanesville, OH	ZAN	90	122	647	51	26	16	50	61
Athens, OH	ATH	88	82	609	53	29	13	69	52
Parsons, WV	PAR	91	50	580	51	28	13	43	56
Uniontown, PA	UNT	94	63	584	48	24	10	46	62
Penn Hill, PA	PEN	90	119	566	49	32	11	80	44
State College, PA	STC	91	97	601	51	31	16	89	30
Transport Region									
Leamington, ON	LEM	93	111	586	51	30	10	54	55
Newtown, CT	NEW	92	50	458	52	37	16	89	38
Egbert, ON	EGB	90	76	618	49	37	13	63	62
Pembroke, ON	PMB	96	55	530	49	33	9	53	64
Dunnville, ON	DUN	89	37	442	50	29	15	51	58
South Brunswick, NJ	SBK	88	131	329	48	42	11	79	46
West Coast									
Simi Valley, CA	SIM	88	129	539	49	31	17	78	40
Livermore, CA	LIV	84	102	582	52	46	18	87	36
Monterey, CA	MTY	90	92	642	52	32	17	91	29
Background									
Springdale, AK	SPG	91	58	506	53	38	13	50	61
Aberdeen, SD	ABD	96	71	676	51	26	11	77	49
Yorkton, SK	YOR	95	82	628	47	39	10	58	58
Penticton, BC	PCT	95	106	621	47	33	13	65	52
All 24 communities		91	2154	13369	50	33	13	67	50

Table 3. Adjusted prevalence of respiratory symptoms by community and symptom: 13,369 children, 24 communities, United States and Canada, 1988–1991[a]

	Asthmatic symptoms (%)				Bronchitic symptoms (%)			
	Asthma	Attacks of wheeze	Persistent wheeze	Any asthmatic symptoms	Bronchitis	Chronic cough	Chronic phlegm	Any bronchitic symptoms
Sulfate Belt								
Hendersonville, TN	3	5	4	7	6	5	2	10
Oak Ridge, TN	5	7	7	10	10	3	2	13
Morehead, KY	5	7	7	9	5	4	3	9
Blacksburg, VA	5	9	9	12	10	5	3	13
Charlottesville, VA	5	8	6	9	9	5	3	12
Zanesville, OH	6	9	7	12	7	6	5	13
Athens, OH	6	8	8	11	7	5	4	12
Parsons, WV	7	7	7	11	7	5	1	11
Uniontown, PA	5	6	7	10	7	5	2	12
Penn Hill, PA	8	10	10	12	9	5	4	14
State College, PA	11	11	12	16	7	9	4	15
Transport Belt								
Leamington, ON	5	7	6	10	4	7	3	12
Newton, CT	6	8	8	11	5	6	3	10
Egbert, ON	9	11	10	14	4	8	5	12
Pembroke, ON	5	8	8	11	4	7	3	11
Dunnville, ON	9	10	10	14	5	8	3	10
South Brunswick, NJ	7	7	7	10	7	7	4	13
West Coast								
Simi Valley, CA	10	10	9	14	7	6	3	12
Livermore, CA	7	8	8	11	3	5	3	9
Monterey, CA	7	8	9	11	8	5	3	14
Background								
Springdale, AR	4	6	6	8	5	5	2	9
Aberdeen, SD	7	9	10	12	8	5	3	14
Yorkton, SK	10	9	9	15	4	4	2	8
Penticton, BC	5	7	6	9	5	4	1	9

[a]Adjusted for sex, history of allergies, parental asthma, parental education, and current smoking in the home. See text for definition of symptoms.

operation, serious chest injury, or heart disease, or who were reported to have received oxygen for more than 2 weeks as newborns were excluded (n = 790). Other exclusions were based on age (not 8–12 years; n = 14), race (not white; n = 1013), and missing information on covariates of interest (n = 337). A total of 13,369 children in 24 communities were included in this analysis.

Overall, half (50%) of the subjects were girls, 33% had a history of diagnosed allergies, 13% had at least one parent with a history of asthma, 67% had at least one parent with some post-high school education, and 50% were currently exposed to environmental tobacco smoke at home. The prevalence of these factors varied by community (Table 2). After adjusting for these factors, the community-specific prevalence of asthma was 3–11%; attacks of wheeze, 5–11%; persistent wheeze, 4–12%; any asthmatic symptoms, 7–16%; bronchitis, 3–10%; chronic cough, 3–9%; chronic phlegm, 1–5%; and any bronchitic symptoms, 8–15% (Table 3).

Particle strong acidity was associated with significantly higher reporting of bronchitis in the past year (Fig. 1 and Table 4; OR = 1.66, 95% CI, 1.11–2.48 for the range of 52 nmol/m^3), as was annual mean sulfate (OR = 1.65, 95% CI, 1.12–2.42 for the range of 7 µg/m^3).

None of the particulate pollutants was associated with significantly higher reporting of asthma, attacks of wheeze, persistent wheeze, chronic cough, or chronic phlegm (Table 4). There was a significant inverse association between chronic cough and PM_{10}. Gaseous acids were associated with a significantly higher risk of asthma (OR = 2.00; 95% CI, 1.14–3.53) and showed a positive association with higher reporting of attacks of wheezing, persistent wheeze, and any asthmatic symptoms (Table 4). Ozone was not associated with a significantly higher risk of any of the respiratory symptoms of interest, regardless of the averaging time used (Table 4). There was a suggestion of a higher risk of chronic cough associated with 24-hr average ozone (OR = 1.29; 95% CI, 0.87–1.91). Ozone for all three averaging times showed some association with higher reporting of bronchitis.

The association between particle strong acidity and bronchitis was similar when the sample was stratified by sex (Fig. 2). The association was sensitive to the inclusion of the Canadian sites, which tended to have low bronchitis prevalence and low to moderate particle strong acidity. Thus, much weaker associations were found when the sample was restricted to U.S. cities or cities studied in the second year, which were predominantly U.S. cities (Fig. 2). No subgroups appeared to be markedly more sensitive to particle strong acidity than the overall sample of children with respect to their risk of bronchitis (Fig. 3). The risk of bronchitis among children who were in the lowest quartile of FVC was not associated with level of particle strong acidity.

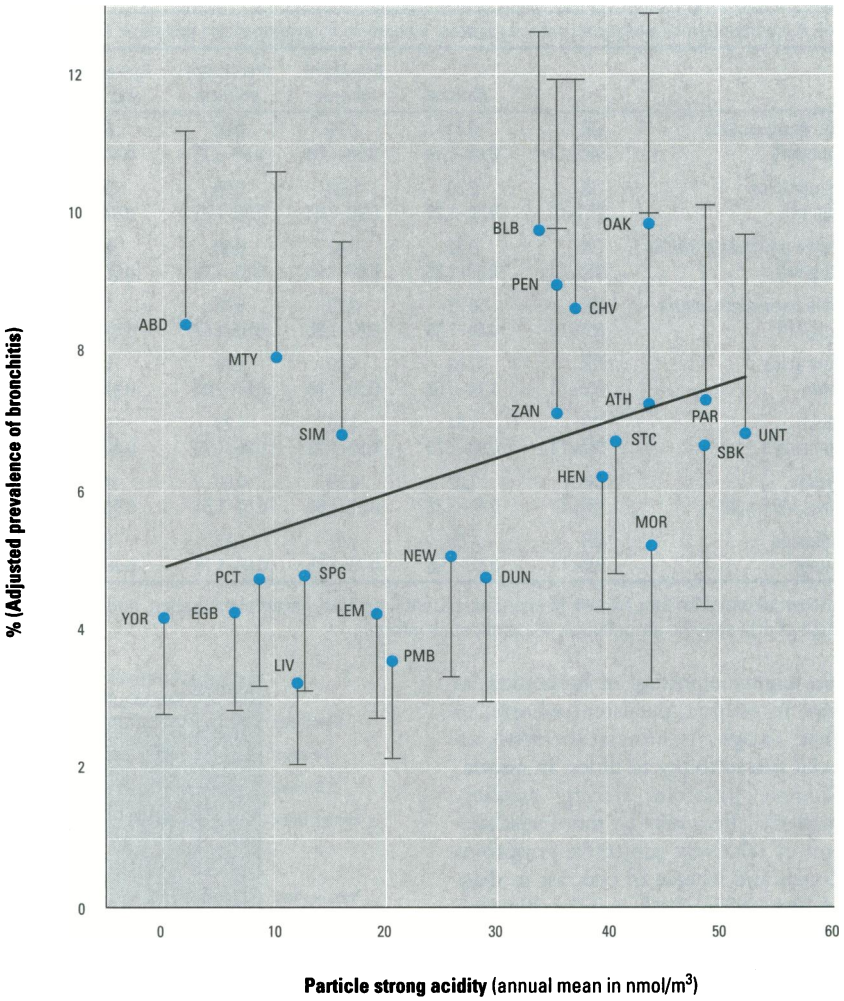

Figure 1. Association of bronchitis with particle strong acidity adjusted for sex, child's history of atopy, parental education, parental asthma, and current smoking in the home: 24 cities, United States and Canada, 1988–1991. One-half 95% CIs for each city-specific bronchitis prevalence are shown. Site codes are provided in Table 2.

Discussion

This study was designed to determine whether long-term exposure to acidic aerosols, estimated by annual mean concentrations, was associated with differences in reported respiratory symptoms in children. The data from this study indicate that adjusted prevalence of bronchitis in children aged 8–12 years was significantly associated with particle strong acidity concentrations. Asthma, persistent wheeze, chronic cough, and chronic phlegm were not significantly associated with higher levels of acid aerosols. No sensitive subgroups were identified.

These results are consistent with earlier reports from the Six Cities Study (*7,8,15*), which noted higher rates of reported bronchitis, but not asthma or wheeze, in association with total suspended particles (*7*), with particulate matter < 15 µm in aerodynamic diameter (*8*), or with levels of particle strong acidity (*15*). In these earlier studies, reporting of chronic cough was higher, but not significantly so, in association with these pollutants.

Prior controlled exposure studies of animals and humans provide support for a relationship between acid aerosols and bronchitis. Short-term exposures to acid aerosols have been shown to result in alterations of mucociliary clearance in rabbits, mice, donkeys, and humans (*24*). Thus it is reasonable to suspect that chronic exposure to moderate levels of acidic air pollution may affect children's nonspecific defense mechanisms adversely, increasing their risk of respiratory illness.

Table 4. Association of air pollutants with respiratory symptoms by symptom and pollutant: 13,369 children, 24 communities, United States and Canada, 1988–1991[a]

		Asthma	Attacks of wheeze	Persistent wheeze	Any asthmatic symptoms	Bronchitis	Chronic cough	Chronic phlegm	Any bronchitic symptoms
Particle strong acidity (52 nmol/m^3)	OR	0.71	0.79	0.83	0.81	1.66	0.92	0.95	1.19
	95% CI	0.43–1.16	0.59–1.06	0.60–1.15	0.60–1.10	1.11–2.48	0.64–1.31	0.60–1.49	0.92–1.53
Sulfate particles (6.8 µg/m^3)	OR	0.80	0.93	0.96	0.91	1.65	1.04	1.29	1.27
	95% CI	0.50–1.29	0.70–1.24	0.70–1.31	0.67–1.24	1.12–2.42	0.74–1.47	0.84–1.96	1.01–1.61
Respirable particulate matter (14.9 µg/m^3)	OR	0.72	0.82	0.80	0.79	1.50	0.82	1.02	1.08
	95% CI	0.40–1.28	0.58–1.15	0.55–1.16	0.55–1.12	0.91–2.47	0.54–1.23	0.61–1.73	0.80–1.46
Inhalable particulate matter (17.3 µg/m^3)	OR	0.77	0.79	0.75	0.75	1.50	0.69	1.05	1.07
	95% CI	0.44–1.35	0.57–1.08	0.53–1.07	0.54–1.05	0.93–2.43	0.48–0.99	0.64–1.72	0.80–1.43
Gaseous acids (2.7 ppb)	OR	2.00	1.40	1.34	1.34	1.18	1.02	1.48	1.11
	95% CI	1.14–3.53	0.99–1.99	0.87–2.05	0.91–1.96	0.67–2.08	0.64–1.61	0.90–2.43	0.80–1.53
Total acid (114 nmol/m^3)	OR	1.64	1.26	1.24	1.24	1.43	0.98	1.40	1.18
	95% CI	0.98–2.73	0.92–1.73	0.86–1.79	0.89–1.72	0.89–2.28	0.66–1.45	0.91–2.15	0.90–1.55
Max. ozone, 1-hr avg. (45.6 ppb)	OR	1.02	0.99	0.90	0.89	1.49	1.13	1.12	1.17
	95% CI	0.44–2.33	0.61–1.60	0.52–1.54	0.53–1.50	0.72–3.12	0.62–2.04	0.53–2.39	0.76–1.81
Sulfur dioxide (12.7 ppb)	OR	1.05	1.07	1.19	1.16	1.56	1.02	1.55	1.29
	95% CI	0.57–1.93	0.75–1.55	0.80–1.79	0.80–1.68	0.95–2.56	0.66–1.58	1.01–2.37	0.98–1.71

[a]Symptoms adjusted for sex, history of allergies, parental asthma, parental education, and current smoking in home. Odds ratio (OR) represents relative risk over the range of city-specific air pollution; CI, confidence interval.

The higher reporting of bronchitis, in contrast to asthma, persistent wheeze, or chronic cough, is consistent with an increased susceptibility to illness in association with particle strong acidity. Alternatively, there may be more misclassification of relatively subjective symptoms like cough and wheeze in contrast to diagnosed bronchitis. Such misclassification may have limited our ability to observe associations of symptoms with air pollution.

Although these data indicate an association between particulate air pollution, specifically particle strong acidity, and bronchitic symptoms, there is no evidence for higher reporting of asthmatic symptoms associated with these pollutants. Similar results in which particulate air pollution was associated with bronchitic symptoms but not with any indicator of asthma have been reported in the Six Cities Study (*7, 8*) for the United States and in German studies (*25*). Children in Leipzig with high sulfur dioxide and particle exposures were compared to children in Munich with much lower exposures to these pollutants. Bronchitic symptoms were elevated in the high sulfur dioxide/particle exposures, but asthmatic symptoms were decreased in Leipzig, the community with higher particle and sulfur dioxide pollution. The authors suggested that asthmatic symptoms were associated with higher ozone concentrations in Munich. However, in the current data the association between asthmatic symptoms and measures of chronic ozone exposure were very slight. No association was found between ozone and respiratory symptoms in the Six Cities Study (*7,8*).

The observed association of asthma and asthmatic symptoms with gaseous acids must be interpreted with caution because this association appears to be driven by only one community. The annual mean for gaseous acid in Simi Valley was one-third higher than the mean for the next highest community (*17*), and Simi Valley also had the second highest rate of reported asthma. If Simi Valley is excluded from consideration, the association between gaseous acid levels and asthma is no longer significant.

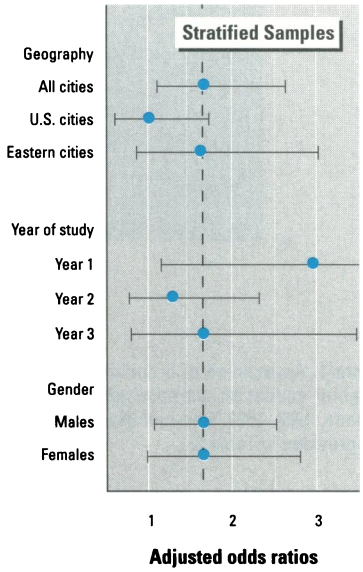

Figure 2. Odds of bronchitis and 95%confidence intervals for a 52 nmol/m^3 increment in particle strong acidity for stratified samples of children or cities adjusted for sex, child's history of atopy, parental education, parental asthma, and current smoking in the home: 24 cities, United States and Canada, 1988–1991. Dashed line indicates overall estimated effect.

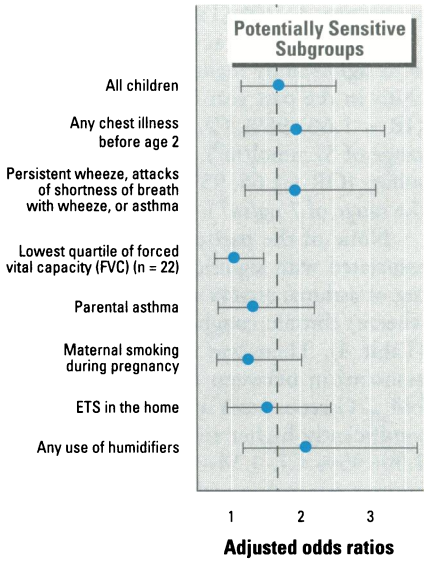

Figure 3. Odds of bronchitis and 95% confidence intervals for a 52 nmol/m^3 increment in particle strong acidity for potentially sensitive subgoups of children or cities adjusted for sex, child's history of atopy, parental education, parental asthma, and current smoking in the home: 24 cities, United States and Canada, 1988–1991. Dashed line indicates overall estimated effect.

There appears to be no evidence that the prevalence of asthma or asthmatic symptoms in children is associated with chronic exposure to particulate, sulfur oxide, or ozone air pollution. This does not imply that acute air pollution episodes do not trigger or exacerbate asthmatic attacks, as has been shown in multiple studies. However, air pollution does not appear to contribute to the increased prevalence of new cases of asthma, as is often claimed in the popular press.

In this study, at least 11 months of air monitoring data were used to calculate annual means of pollutants as a surrogate for lifetime exposure. The validity of such a monitoring scheme depends on the representativeness of the selected year's aerometrics. Brook and Spengler (*26*) have examined historical aerometric data and concluded that community air pollution levels have been relatively stable over these children's lifetimes and that the relative rankings of the communities with respect to particle strong acidity, ozone, and PM_{10} were not likely to have changed. Although it is possible that the children differed in their personal activity and exposure patterns between communities, these differences were not likely to be related to annual means of acid air pollution.

Particle strong acidity, sulfate, and fine particulate matter are all formed in secondary reactions of the emissions from fossil fuel combustion. They have similar regional and temporal distributions. The strong correlations of several pollutants in this study, especially particle strong acidity with sulfate (r = 0.90) and $PM_{2.1}$ (r = 0.82), make it difficult to distinguish the agent of interest. This study was designed to have a low correlation of particle strong acidity with ozone, and although high ozone was positively associated with reporting of bronchitis, the association was not statistically significant.

The sampled children in each community were selected to have a narrow range of age and race. Communities also were selected to have similar demographic characteristics. We adjusted for differences among communities in the distributions of sex, child's allergy history, parental education, parental asthma, and current smoking in the home in the first stage of the two-stage model. No evidence of effect modification was observed among these children. The association of bronchitis with particle strong acidity was similar regardless of sex, year of study, country, home characteristics, or child's health history. The only exception was for children in the lowest quartile of FVC, who had no association between a higher risk of bronchitis and particle strong acidity.

These data indicate that chronic exposure to acid aerosol pollution may have observable negative consequences for the health of children. Although the long-term consequences of bronchitis in these children remain unclear, respiratory illnesses in childhood may be a risk factor for chronic obstructive disease. These children also may be at a higher risk from other environmental and occupational exposures later in life.

References

1. American Thoracic Society. Health effects of atmospheric acids and their precursors. Am Rev Respir Dis 144:464–467 (1991).
2. U.S. EPA. An acid aerosols position paper: health effects and aerometrics. ECAO-R-0140. Research Triangle Park, NC:Environmental Protection Agency, 1987.
3. Graham JA. Review, discussion, and summary: toxicology. Environ Health Perspect 79: 191–194 (1989).
4. Gong H. Health effects of air pollution. A review of clinical studies. Clinics Chest Med 13:201–214 (1992).
5. Dassen W, Brunekreef B, Hoek G, Hofschreuder P, Staatsen B, deGroot H, Schouten E, Biersteker K. Decline in children's pulmonary function during an air pollution episode. J Air Pollut Control Assoc 36:1223–1227 (1986).
6. Graham NMH. The epidemiology of acute respiratory infections in children and adults: a global perspective. Epidemiol Rev 12:149–178 (1990).
7. Ware JH, Ferris BG Jr., Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particulates on respiratory health of preadolescent children. Am Rev Respir Dis 133:834–842 (1986).
8. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. Am Rev Respir Dis 139:587–594 (1989).
9. Bates DV, Baker-Anderson M, Sizto R. Asthma attack periodicity: A study of hospital emergency visits in Vancouver. Environ Res 51:51–70 (1990).
10. Pope CA III. Respiratory hospital admissions associated with PM_{10} pollution in Utah, Salt Lake, and Cache Valleys. Arch Environ Health 46:90–97 (1991).
11. Ostro B. The association of air pollution and mortality: Examining the case for inference. Arch Environ Health 48:336–342 (1993).
12. Lippmann M. Background on health effects of acid aerosols. Environ Health Perspect 79:3–6 (1989).
13. Spengler JD, Brauer M, Koutrakis P. Acid air and health. Environ Sci Technol 24:946–956 (1990).
14. Spengler JD, Keeler GJ, Koutrakis P, Ryan PB, Raizenne M, Franklin CA. Exposures to acid aerosols. Environ Health Perspect 79:43–51 (1989).
15. Damokosh AI, Spengler JD, Dockery DW, Ware JH, Speizer FE. Effects of acidic particles on respiratory symptoms in 7 US communities. Am Rev Respir Dis 147:A632 (1993).
16. Speizer FE. Studies of acid aerosols in six cities and in a new multi-city investigation: design issues. Environ Health Perspect 79:61–67 (1989).
17. Spengler JD, Koutrakis P, Dockery DW, Raizenne M, Speizer FE. Health effects of acid aerosols in North American children: air pollution exposures. Environ Health Perspect 104:492–499 (1996).
18. Marple VA, Rubow KL, Turner W, Spengler JD. Low flow rate sharp cut impactors for indoor air sampling: design and calibration. J Air Pollut Control Assoc 37:1303–1307 (1987).
19. Koutrakis P, Wolfson JM, Slater JL, Brauer M, Spengler JD, Stevens RK. Evaluation of an annular denuder/filter pack system to collect acidic aerosols and gases. Environ Sci Technol 22:1463–1468 (1988).
20. Koutrakis P, Wolfson JM, Spengler JD. An improved method for measuring aerosol strong acidity: results from a nine-month study in St. Louis, Missouri and Kingston, Tennessee. Atmos Environ 22:157–162 (1988).
21. Keeler GJ, Spengler JD, Castillo RA. Acid aerosol measurements at a suburban Connecticut site. Atmos Environ 25A:681–690 (1991).
22. Keeler GJ, Spengler JD, Koutrakis P, Allen GA, Raizenne M, Stern B. Transported acid aerosols measured in southern Ontario. Atmos Environ 24A:2935–2950 (1990).
23. Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, Ware JH, Speizer FE. Health effects of acid aerosols on North American children: Pulmonary function. Environ Health Perspect 104:506–514 (1996).
24. Schlesinger RB. The interaction of inhaled toxicants with respiratory tract clearance mechanisms. Crit Rev Toxicol 20:257–286 (1990).
25. Von Mutius E, Fritzsch C. Prevalence of asthma and allergic disorders among children in United Germany; a descriptive comparison. Br Med J 6866:1395–1399 (1992).
26. Brook JR, Spengler JD. Exposure to fine particle acidity and sulfate in 24 North American communities: the relationship between single year observations and long-term exposures. Air Waste Manage Assoc 45:709–721 (1995).